it is absolutely necessary that you should be well acquainted with the general relation of these structures to one another before attempting to investigate the early condition of the embryo. Let me tell you that a knowledge of the relation of the fœtus to the uterus, even so far as is necessary for the practice of midwifery, is not to be obtained in any other way than by direct observation; and I can assure you that this kind of knowledge is well worth the trouble of obtaining once for all, for it will enable you to treat the various accidents which arise in the course of pregnancy with far greater readiness and success than if you trust alone to acquaintance with written authorities on the subject.

ON THE

IMMEDIATE CAUSES OF THE CHANGES OF THE HEART AS PRODUCED IN ENDOCARDITIS.

BY WALTER MOXON, M.D., F.R.C.P.

Inflammation; distribution of endocarditis. — It is well known that inflammations of the heart are by far most frequent on its left side. Indeed endocarditis of the right side is exceedingly rare, except in cases of malformation. In examinations of the heart it must also be remembered that the endocardium of the left heart is thicker than that of the right. It is further true that fibroid patches, syphilitic or otherwise, occur most frequently on the left side, so also do the fatty changes, both that occurring in streaks, and that permeating the whole thickness in the overworked hearts of aged people. Thus I may say that the strongly-acting and hard-worked ventricle suffers most disease. I shall go on presently to show that the seats where endocarditis declares itself in the left side of the heart are almost invariably places where friction is exerted, and I shall have to point out that the rarer cases where endocardial thickening and inflammation occur in the right heart are always cases of unusual hypertrophy of the right heart, where it exerts more mechanical power, and therefore causes more wear and tear of the structures connected with it. All this evidence accumulates to prove that the reason why the left heart suffers more than the right is because it is stronger and works harder, straining the mechanism of its valves, and irritating its lining membrane with the friction of the blood upon it; while the right heart escapes because its play on its contents and valves is less forcible. As a further proof of this, we must remember that in fœtal life, when the right heart is doing all the work, any disease almost always attacks the right side of the heart.

General characters of endocarditis —When I speak of inflammation of the endocardium or endocarditis, it must not be thought that any such process ever occurs all over the endocardium, as pericarditis is found all over the pericardium, or peritonitis all over the peritoneum. That is, we never find the whole endocardium even of a single cavity coated with lymph or other products of the inflammation. The inflammation is *always circumscribed.* We may occasionally find several such patches of endocarditis discoverable by granulations or fibrin on the membrane, but, except in the rarest cases, these patches are within reach of a fibrinous clot on a valve, which no doubt struck the affected part in the action of the heart. When considering the possibilities of such contact in any specimen, we must bear well in mind how the heart closes its cavity in contracting, and thus brings together parts that in the dilated and dead heart are remote from each other. The study of a great number of cases has led me to conclude that *such friction with fibrin clots, together with mechanical strain, make the principal, if not the sole, direct cause of endocarditis;* rheumatism and other general states creating only a vulnerability of the fibrous structures, so that they cannot resist the irritation of the friction. As a corollary from these facts, it follows that we must in all cases where endocarditis is suspected do all in our power to moderate the force of the heart's action, so as to place the left heart as much as possible in the same condition as the right, reducing the friction which I have nearly proved to be the sole efficient cause of the anatomical changes that result from endocarditis.

Comparison of endocarditis with other inflammations.—When inflammation was regarded as an act proper to the vessels, and when it was doubtful whether the endocardium had any vessels, it was naturally a rather perplexing question which inquired whether the apparently inflammatory effects of endocarditis were really due to inflammation. The difficulty was much greater in the case of endocardium than in other vascular structures, such as the cornea or cartilage, which produce little or no inflammatory product, because it was easy to deny in these the existence of inflammation in the apparent absence of its results; but a great quantity of lymph-like deposit was seen in endocarditis, and hence arose a rather keen discussion, some being disposed to think this deposit was really formed from the endocardium, as lymph is produced by other inflamed serous surfaces, while others thought the apparent lymph was only fibrin of the blood which had precipitated itself, and which was known to be in excess in rheumatism, the disease that commonly causes endocarditis. The following out of this question by Lee and others led to some interesting experiments on the inflammability of the inner vascular surfaces, and it was proved by them that the endarterium will not produce lymph and pus freely on its surface, like an ordinary serous membrane.

But the great number of more careful microscopic examinations which have been made of late have settled this question decisively, although with some alteration of the standpoint from which it is viewed. This alteration arises from the fact that inflammation is no longer regarded as *exclusively* the act of the vessels, although the actions in the vessels compose its chief and most obvious phenomena in ordinary inflammations.

It was clearly shown by Virchow and others that a more constant and perhaps the essential act in inflammation is one of irritation in the parenchymatous parts of the textures as represented by their cellular elements. With this belief it has become no longer possible for any to hold that endocarditis cannot occur on the score that the valves are evascular, while it is further proved that some at least of the valves — certainly the mitral — do contain capillary vessels which have been observed to be congested in inflammation. Then at present we are able to follow out the inquiry into the process of endocarditis on the same terms as we examine the inflammations of other tissues so far as the structure of the endocardium is concerned. But it remains true that its circumstances are very peculiar in that it exposes its large surface to contact with the blood. For the living blood is proved by observations and experiments to be always ready to deposit fibrin on a roughened surface, and especially so when the fibrin is in excess in the blood, and when the blood is arterial, and when the blood is in contact with a surface of no vitality or low vitality, and when its current is checked. Now, all these conditions are found in the inequalities of an inflamed endocardial surface, and especially when the fibrinous deposit has already commenced on several adjacent spots, for there is then a rough surface of low vitality retarding the current in its inequalities, the blood from inflammation being hyperinotic, and the left heart containing arterial blood. Recognising these conditions, one is prepared to believe that any change in the endocardium inducing a roughening of its surface by swelling or exudation would soon lead to the deposit of concretions on the roughened parts. Such a deposition undoubtedly occurs, and it is this lodgment of fibrin in quantities on the inflamed surface that constitutes the peculiarity of endocarditis and causes it to differ from inflammation in all other parts. It is a most unfortunate thing that in the heart, where their consequences must be so exceptionally unhappy, the effects of inflammation are so permanent; and we may ask why it is that when rheumatism affects the joints and the heart only equally severely, the former should so commonly recover their integrity while the latter is permanently damaged. The permanence of the injury in the case of endocarditis is simply due to the want of counter-pressure. In the joints the swollen membranes are pressed against other solid structures as soon as the liquid effusion is removed; this pressure causes absorption of all the new products: whereas in the heart there is no direct pressure of solids against the inflamed

valves, which stand freely bathed in fluid blood, so that the new products persist.

Special characters of endocarditis.—Endocarditis is either plastic or ulcerative. Let us consider first the characters of its plastic form. When found in its earliest stage it appears as a slight swelling, with sometimes a pink colour of the membrane. (As to this colour I am not certain whether it can be regarded as due to congestion or imbibition of colouring-matter.) This always occurs near the edges of a valve in the formation of a line of little elevations along the contact line of its segments, where the friction is greatest. Some have thought that this is due to a peculiarity in the composition of this contact line of the valves. It is true that this line is usually more fibrous and thick than the rest of the valve; but this fibrous thickness itself is clearly due to the chronic irritation of that line in the action of the heart. For, first, it is not found in young subjects, and is thicker as age increases; secondly, it is not found in the valves of a normal right heart; and, thirdly, it is found in the valves of the right heart when that heart is hypertrophied. A row of little elevations along this line is what we find in chorea, in acute rheumatism, in puerperal pyæmia, &c.—that is, generally in acute plastic endocarditis, when the change in the heart is quite early. Such a change may give rise to a soft bruit in the action of the heart, but it cannot much obstruct its orifices, nor can it poison the blood with its products, so that at this stage it is of little importance; it is only its after-consequences that are grave. I have already pointed to the absence of counter-pressure as causing permanence of the swelling of the valve; and hence it is that the heart, after acute rheumatism, chorea, &c., exerts its strain on an unrecovered, thickened, and softened structure. It is curious to observe how constantly we find in all cases of endocarditis from chorea and acute rheumatism, pyæmia, &c., when death occurs during the acute illness, that the change in the valves is limited to this line of bead-like elevations along the meeting edges of the segments. If a valve with these nodules be cut for the microscope across the plane of its curtain, so as to show a section down through one of the small nodules, this will be found to be composed of a simple cloudy swelling of the tissue of the valve, with a multiplication of the cellular elements in its fibrous structure raising its surface into a little hillock. But if the nodule is in the form of a distinct projecting grain, there will always be found on the top of this hillock a cap of fibrin, separated from its substance by a line which the microscope defines very clearly. This cap of fibrin differs in composition from the hillock itself, though the difference is more easily seen than described, for the organisation in both is very low; but the fibrin is almost structureless, while the hillock of swollen valve-tissue shows the regularly placed nuclei of fibrous tissue. Specimens of endocarditis in this early stage are frequent enough; but it is not easy to say what occurs next in the process, because we do not have many opportunities of seeing the intermediate conditions between this which is found in cases of death from the acute disease of which the endocarditis is a part and that advanced state of change in the valves which long afterwards proves fatal by disabling them. It appears to me that, in the interval between acute endocarditis of rheumatic fever and the death long after from valvular disease of the heart, many frequent repetitions of the inflammation must occur. Sometimes we find inflammatory products of two dates on the valves—some recent, some older. But no doubt a constant state of inflammatory irritation remains, slowly changing the valve. The cause is probably this, that the valve remains swollen through absence of the pressure on it which is required to restore it, as I have already said, and being thus unable to return to its natural size, and being still softer than natural, it is both subjected to more friction and less able to resist the effects of this constant action of the heart, keeping up a chronic irritation of the unhealed valve, which gradually leads to great thickening of the valve, with a scar-like tissue in which calcareous salts are often deposited, the whole causing those miserable effects in contraction and deformity with which we are too familiar. Here, again, I would insist upon the necessity of warning all persons who have had rheumatic fever or chorea against such muscular exertion as will greatly increase the action of the heart and the friction of its valves.

The *ulcerative* form of acute endocarditis always begins in a valve. It must not be supposed to usually accompany the milder plastic form we have just described. Some authorities proceed in their description of endocarditis as if, when the swelling of the membrane occurs, the next thing usually is for this swelling to break down into an ulcer. But we must not let such an impression mislead us into thinking that ulcerative endocarditis is an ordinary part of common cases of endocarditis, such as we meet in chorea, rheumatism, &c. The occurrence of ulceration is a rare and formidable complication of plastic endocarditis. When ulceration is limited to the endocardium—i.e., when it is only in a valve—an abrasion of the inflamed surface forms, and the affected side of the valve suffers a breach. The valve is, as you are aware, composed of two layers of endocardium, with some fibrous tissue and a few vessels between them. Now when one layer of the endocardium is breached by the ulcer, the force of the heart drives the blood into the hollow, and presses before it the remaining layer of the endocardium, thus forming an acute aneurism of the valve. Such an aneurism of course projects away in the direction of greatest pressure, so that in the mitral valve it bulges up into the auricle in the aortic valves downwards into the ventricle. It is but too easy then for the ulceration or the heart's action to work through the remaining layer of endocardium, and so perforate the valve. Such perforations are not very infrequent either in the mitral or the aortic. They are always covered all around with "vegetations"—i.e., nodular masses of fibrin,—which hide the opening. These vegetations may reach a large size and become calcareous, and by friction start ulceration in the wall of the heart where they come in contact with it. Such ulcers and perforations of the valves, with the ulcerations of the muscle of the heart which are apt to extend from them, constitute a dangerous disease, having characters quite distinct from the plastic form of endocarditis, which is only dangerous through its subsequent effects in causing contraction of the valve, and so inducing dropsy &c. Ulcerative endocarditis is generally accompanied by pyrexia. It may produce pyæmic suppurations by embolism of distant organs with particles from the ulcer, or the large fibrinous masses around the ulcer may move off and plug the cerebral or femoral arteries &c., causing hemiplegia, gangrene of foot, or other severe lesions, through simple obstruction. Ulceration may supervene on chronic plastic endocarditis and its symptoms, and the cases be complicated with the obstructive effects of this. But it usually kills without dropsy. Sometimes in its typical form I have known it mistaken for continued fever. Some say that pus may be found as little abscesses in the tissue of the valves under these circumstances, but of this I have no experience.

Limitation of endocarditis to the valves and their neighbourhood.—I would repeat that all I have said as to acute endocarditis applies almost solely to the valves, and to such extension from the valvular changes as arises through the spread of the ulcers to the attachments of the valve, or the friction of masses of fibrin on the neighbouring parts. Very occasionally we find a patch of nodular thickening of the endocardium in the left auricle, about the root of the mitral, and once or twice, under exceptional circumstances, over a partial rupture of the septum ventriculorum.

If it be asked what practical good is to arise from these facts and reflections, I should, in reply, express a hope that we should more generally recognise that the chronic obstructive heart disease which follows rheumatism is due to friction of the slowly-healing valves, and that it is a slow and continuous process. Knowing this, the indication pointing out the necessity of prolonged rest after rheumatic fever is sufficiently evident; indeed every measure must be taken to ensure that the action of the heart be as gentle and quiet as possible.

BEQUESTS &C. TO MEDICAL CHARITIES.—The Rev. Charles Ingleby, of Wood Bank, Cheadle, bequeathed £100 each to St. George's Hospital, the Royal Asylum for Incurables, Putney, and the Earlswood Asylum for Idiots, and £50 each to the General Hospital, the Queen's Hospital, and the General Dispensary (all at Birmingham), the North Staffordshire Infirmary, the Brompton Hospital for Consumption, the National Cottage Hospital, Ventnor, and the Hospital for Sick Children, Great Ormond-street. St. Mary's Hospital, Paddington, has received £105 from Mr. Henry J. Gardiner.